

Differences in *Acinetobacter baumannii* Strains and Host Innate Immune Response Determine Morbidity and Mortality in Experimental Pneumonia

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Titre Differences in *Acinetobacter baumannii* Strains and Host Innate Immune Response Determine Morbidity and Mortality in Experimental Pneumonia

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Despite many reports documenting its epidemicity, little is known on the interaction of *Acinetobacter baumannii* with its host. To deepen our insight into this relationship, we studied persistence of and host response to different *A. baumannii* strains including representatives of the European (EU) clones I-III in a mouse pneumonia model. Neutropenic mice were inoculated intratracheally with five *A. baumannii* strains and an *A. junii* strain and at several days morbidity, mortality, bacterial counts, airway inflammation, and chemo- and cytokine production in lungs and blood were determined. *A. baumannii* RUH875 and RUH134 (EU clone I and II, respectively) and sporadic strain LUH8326 resulted in high morbidity/mortality, whereas *A. baumannii* LUH5875 (EU clone III, which is less widespread than clone I and II) caused less symptoms. *A. baumannii* type strain RUH3023T and *A. junii* LUH5851 did not cause disease. All strains, except *A. baumannii* RUH3023T and *A. junii* LUH5851, survived and multiplied in the lungs for several days. Morbidity and mortality were associated with the severity of lung pathology and a specific immune response characterized by low levels of anti-inflammatory (IL-10) and specific pro-inflammatory (IL-12p40 and IL-23) cytokines at the first day of infection. Altogether, a striking difference in behaviour among the *A. baumannii* strains was observed with the clone I and II strains being most virulent, whereas the *A. baumannii* type strain, which is frequently used in virulence studies appeared harmless.

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